BEDSIDE MEDICINE FOR BEDSIDE DOCTORS

An open forum for brief discussions of the workaday problems of the bedside doctor. Suggestions for subjects for discussion invited.

ANEMIA

JOHN MARTIN ASKEY, LOS ANGELES.—Anemia properly exists when there is an absolute reduction in the total blood quantity in proportion to the body weight. Normally, there are about eighty-three cubic centimeters of blood for each kilogram of body weight. Anemia may exist with a normal erythrocyte count and a normal hemoglobin determination as in true oligemia such as immediately follows an acute hemorrhage.

Extreme dehydration by concentration may change an anemic blood picture into a normal or even polycythemic blood picture. These conditions are usually transitory, however, and ordinarily the true blood picture is reflected accurately by the determination of the erythrocyte count, the hemoglobin percentage and the study of the blood smear. Further blood studies should be made as indicated by clinical evidence and suspicion.

The actual blood picture at any time is the resulting quotient of the rate of blood regeneration and the rate of blood loss, whether by hemorrhage or hemolysis. Clinically, classification according to derangement of the function of blood formation or disturbance of the normal function of destruction, or actual loss through hemorrhage seems most helpful.

Minot's classification is simple but adequate.

1. Anemias due to blood loss.

2. Anemias partially dependent or wholly dependent on defective blood formation.

3. Anemias in which blood destruction is usually marked and where anemia is partially or wholly dependent on abnormal blood destruction.

Most of the anemias can be classified under one type, but many overlap. Blood loss may occur to complicate anemias essentially due to defective formation or increased destruction. Thus a patient with typical pernicious anemia may develop bleeding hemorrhoids.

In idiopathic aplastic anemia the anemia is primarily due to failure of formation of the red cells, due to depression first of the erythropoietic centers of the bone marrow. At a later stage, the platelet centers are involved, the platelets drop, and mucous membrane hemorrhages develop to further increase the anemia.

Conversely, anemia due to chronic blood loss such as results from menorrhagia, an oozing peptic ulcer, or bleeding hemorrhoids, may so tax the regenerative powers of the bone marrow as to produce a secondary depression with a reduction in blood formation.

I shall consider only the *anemias due to blood loss*. These may be acute or chronic anemias.

A massive loss of blood causes at first a true oligemia with no change in the blood count. With later blood volume restoration, anemia is evidenced by the reduced red cell count and lowered hemoglobin, with greater proportionate lowering of the hemoglobin. The bone marrow response is marked, and rapid proliferation occurs of the three cellular elements, the polymorphonuclear leucocytes, the platelets and the red cells. The polymorphonuclear leucocytosis may rise to thirty or forty thousand with a platelet count of one million and a reticulated red cell percentage as high as ten. The red cells are young with blasts present.

In an anemia due to chronic blood loss of small amounts, the bone marrow response is much reduced, with consequently only slight leucocytosis, moderate platelet increase and slight increase in the percentage of reticulated red cells. If the blood loss be unchecked, compensation by bone marrow hyperplasia may fail and a hypoplastic type of anemia result.

In the study of spontaneous hemorrhage from any site, be it epistaxis, oozing gums, melena, hematemesis or purpura, it is of primary importance to determine the status of the blood coagulation factors. The first sign of a hemorrhagic purpura, leukemia or other blood dyscrasia may be slight mucous membrane hemorrhage. One patient with incipient aplastic anemia due to benzol poisoning developed oozing of the gums. Severe pyorrhea was thought to be the cause and a tooth was extracted with disastrous hemorrhage.

Another patient first noticed petechial spots below the line of constriction of the garters on his calf. In a few days, there developed epistaxis and oozing of the gums. Blood study revealed an acute leukemia.

In addition to a careful study of the blood smear and a white cell count the determination of (1) the bleeding time, (2) the clotting time, (3) the platelet count, (4) the retractility of the clot and (5) the Rumpel-Leed tourniquet test for decreased capillary resistance usually will explain the cause of pathologic hemorrhage.

Treatment of anemia from blood loss should be directed at control and correction of any organic lesion, at correction of any blood coagulation disturbance, and stimulation of hematopoiesis by diet and iron therapy.

Most spontaneous pathologic hemorrhages are due to platelet deficiency and are best controlled by supplying that deficiency by whole blood. This is a temporary benefit only, of course, as the life of the platelet is only three to five days. Repeated transfusion, however, may tide the body over a crisis until regeneration occurs, as in the case of a secondary aplastic anemia, or it may be life

saving in preparation of a patient with chronic hemorrhagic purpura for splenectomy. In the acute leukemias it is merely a palliative procedure.

The anemias due purely to chronic blood loss are helped often by whole liver feeding. Liver extract has proved far inferior to the whole liver. Although the response is often slow, kidney, beef heart, muscle meat and certain fruits as peaches, apricots and prunes are definitely effective.

Paradoxically, spinach has been proved of little value.

Iron is valuable in chronic anemia due to blood loss where the cause has been removed. It obviously is ineffectual where hemolysis is the cause, or where blood loss is slowly continuous. Given in adequate, large doses (equivalent of one gram daily of metallic iron) in properly selected cases, iron is a valuable adjunct, particularly if supplemented by whole liver.

* * *

STACY R. METTIER, SAN FRANCISCO.—*Anemia particularly dependent or wholly dependent on defective blood formation* is usually chronic in nature and as a rule results from chemical, toxic or mechanical alteration of the bone marrow. In this group may also be included those anemias which result from imbalance of endocrine control and others due to a deficiency in some element essential to the maturation of red blood cells. Among the bone marrow intoxications are those resulting from poisoning by lead, mercury and arsenic, and intoxications resulting from tissue alteration such as occur in the kidney with nephritis or from the liver with cirrhosis. Mechanical alteration of the bone marrow may result from tumor metastases with a crowding out of the elements essential to blood formation. This is called myelophthisic anemia.

It has long been known that inadequate diet may result in the production of anemia. This is especially true in the rather vague and unexplained anemias occurring in females. Among the anemias of this type is one originally described by Faber, which is associated with a gastric achlorhydria and in some instances atrophy of the mucous membrane along the lateral margins of the tongue. This anemia is of the secondary type with a low color index. It is essentially an iron deficiency anemia and will respond dramatically to large doses of iron.

Our views concerning pernicious anemia have been greatly changed through the epoch-making contribution of Castle on the etiological relationship of achylia gastrica to the anemia of this disease. This investigator has shown that the disease is virtually a deficiency disease of a relatively new type. He states there is a lack in the secretion of the gastric mucosa of an element he calls the intrinsic factor. This intrinsic factor he has shown is neither the hydrochloric acid nor pepsin. He states further that this intrinsic factor is essential for the elaboration from beef protein of the antipernicious anemia substance. This substance he calls the extrinsic factor and is, he believes, probably nitrogeneous in nature. We now know through the contributions of Minot and Murphy that this antipernicious anemia substance is present in liver and kidney and that a dried extract of the liver is effective when used in adequate amounts in the amelioration of the anemia. Minot and Cohn and West working independently have also prepared an extract which when administered intravenously is also effective in the treatment of this disease. Peabody has shown that this antipernicious anemia substance causes a decrease in the number of megaloblasts in the bone marrow and that there occurs an increasing number of normoblasts. Thus the substance permits maturation of the red blood cells.

Aplastic anemia is also included in this group of anemias due to defective blood formation. There is a type originally described by Ehrlich in 1888 in which the etiological factor is unknown. This disease may be either acute or chronic in course with the development of a profound anemia, a leukopenia and petechial hemorrhages. The outcome is usually fatal. An aplastic type of anemia may be induced by certain toxic agents especially benzol and arsphenamin preparations.

The anemia associated with certain vitamin deficiencies such as occur in scurvy or pellagra may also be included in this group. The anemia of this type may be readily alleviated with the ingestion of large amounts of the appropriate vitamin.

* * *

HARRY WYCKOFF, SAN FRANCISCO.—The third form of anemia, or more properly, group of anemias in Minot's Classification, is defined as: *"Anemia in which blood destruction is usually marked and where the anemia is dependent partially or wholly on abnormal blood destruction."*

That the forms of anemia included in this group are in fact anemias largely, or apparently entirely the result of excessive (abnormal) blood destruction is indicated by clinical and laboratory evidence of blood destruction in excess of normal, and further by evidences of corresponding regenerative response on the part of the bone marrow.

Some of the conditions included in this group are in part due to marrow deficiency but the converse is true in regard to some of the anemias included in the second group in this classification. As Doctor Askey has pointed out in his discussion, types may overlap.

I. Pernicious anemia. Diagnosis rests upon three general groups of findings: Blood changes, neurological signs, gastro-intestinal symptoms and signs. Important are: Papillary atrophy of the tongue; gastric anacidity (present even after histamin stimulation); impaired vibratory sense (legs); splenic enlargement. Findings in the blood may show considerable variation dependent upon the stage of the disease.

II. Conditions in which the anemia is usually of the simple type but which may sometimes exist as chronic anemia of the hemolytic type.

Examples are Bothriocephalus latus infestation, malignant tumors (particularly of the stomach), lues (which most often produces anemias of the simple chronic type).

III. Acute hemolytic forms of anemia in which the red cell count tends to fall rapidly so that the color index of the blood is usually relatively high. Regenerative activity is marked so that qualitative changes in the red cells are evident and immature leucocytes appear in the peripheral blood. (Leukemia.)

Anemias of this kind may occur in infections as with streptococcus, in malaria, after severe burns, in poisoning as with phenol, potassium chlorate, acetanilid, aniline.

IV. Chronic forms of hemolytic anemia. Hemolytic jaundice (acholuric family jaundice) in which the essential features are, anemia with signs of hemolysis, splenomegaly, jaundice from an early age, and abnormal fragility of the red cells.

There appears to be an acquired type of the disease in which the anemia predominates the jaundice in the picture and there is a suggestion of relationship to splenic anemia (Banti's Disease). This resemblance of splenic anemia cases to some cases of hemoyltic jaundice, the occurrences of evidences of blood destruction (urobilinuria), and the improvement of symptoms in cases of both types after splenectomy suggests that splenic anemia might properly be included in the group of hemolytic anemias in this classification.

It is obvious that adequate discussion of the treatment of this important group of anemias cannot be undertaken in any limited space. In general the treatment rests upon three principal measures, liver therapy (with special reference to pernicious anemia), transfusion, and splenectomy in selected cases.

The Monastic Infirmaries.—The infirmary of the medieval monastery, with special reference to the Abbey of Westminster, was the subject of a lantern lecture delivered by Mr. Percy Fleming, emeritus professor of ophthalmic medicine and surgery, at University College, London, on February 21. The lecturer stated that there was no infirmary belonging to a monastery in this country standing at the present time; all that remained were some well-preserved ruins. But it was possible to reconstruct in the imagination the character of the monastic infirmary in the Middle Ages, and from the ruins of Furness Abbey in particular a great deal was to be learned with regard to the domestic offices of the monks. Generally speaking, the west end of a monastery was used for business purposes, and the east cloister was the site of the infirmary. Those who designed the monasteries were not unfamiliar with the principles of sanitation, and much thought was given to the lay-out and slope of the ground with a view to drainage. Attached to the monastery was always a garden devoted to the growing of herbs for medicinal purposes. Coming to Westminster Abbey, Mr. Fleming drew attention to the fourteenth century door in the middle of the east wall in the Little Cloisters; this, he said, was the door of the infirmary chapel, rebuilt after a fire. Incidentally, a very common ailment among the monks was disease of the shin bone, possibly brought about through much kneeling at a time before hassocks were available. It might easily have originated as a small abrasion, and as baths were very infrequent—at Westminster the allowance of baths was only two a year—possibly the neglected abrasion developed into more serious trouble. It was recorded at Westminster that in 1310 one monk had the trouble in both legs, and another entry, under the date 1334, stated that a monk had surgical treatment for it at a total cost of forty-six shillings. Bleeding was a common form of treatment; there was always a group of people in the monastic infirmary who had recently been bled. Even apart from any illness, there was periodic bleeding in those days, and after it a certain amount of relaxation was allowed to the monks. The records seemed to show that they had a good time in the infirmary, and were anxious rather than otherwise to be bled, even more often than was necessary. The dean of Westminster, who presided at the lecture, spoke in appreciative terms of the perfection of the arrangements of the modern hospital, and the blessings which scientific medicine and surgery had brought. The difference between the treatment now available to the poorest and that which was given in the old times in the monastic infirmary was so immense that the two things could hardly be spoken of in the same breath. He added that at Westminster Abbey the infirmary was used for various purposes, not limited to the care of the sick or the convalescent. It was there that the young monks were regularly whipped, the idea apparently being that the place where health was restored to the body was the best place for correcting the soul. It was in the infirmary also that the "playfellows" were housed. These were monks of over fifty years' standing, who were constituted censors of everything that went on in the monastery. It might be imagined, said Dr. Foxley Norris, how nice a state of things existed when a party of playfellows—a most inappropriate name—lived at their ease in the best quarter of the monastery, their only occupation being to criticize the others. But the debt of medical science to the monasteries must not be forgotten. The monasteries were at the beginning of nearly everything. Whatever poor relief was distributed in those days came through the monastic infirmary. The history of England on the social side might be described as the gradual taking over by the public authority of what in its origin was voluntary and vouchsafed in the name of religion. But he trusted that the day was far distant when the voluntary hospital, which represented the spirit of the old monastic infirmary, but represented also the best that modern science and skill could command, crossed the same Rubicon.—*British Medical Journal,* 1, 406, March 1, 1930.

An Honor to Dr. Harvey Cushing.—The medical profession will be especially pleased to note the award of the Montclair Yale Bowl for 1930 to Dr. Harvey Cushing by the Yale Club of Montclair, New Jersey. The presentation will be made December 6 at the tenth annual barn party following a custom of the past decade which has recognized eminent graduates of Yale at intervals. In naming Doctor Cushing, the committee stated:

"We feel that Doctor Cushing is eminently entitled to receive this award which each year goes to a Yale alumnus who, by his own efforts, has made his "Y" in life. Doctor Cushing is recognized in medical circles as one of the most outstanding of modern surgeons. He graduated from Yale in 1891 and has received two honorary Yale degrees since that time, one in 1913 and one in 1919. He is chief surgeon at the Peter Bent Brigham Hospital, Boston, and is the author of many books, including the "Life of Sir William Osler," which won him the Pulitzer prize.

"We have further selected Doctor Cushing as the recipient of the award this year because we have felt that it would be fitting to bestow the bowl upon a Yale alumnus who has made his "Y" in scientific and professional fields. The award has not previously gone to any professional man or scientist, although Yale alumni include many distinguished leaders in these walks of life."

It is gratifying to know that the achievements of an eminent surgeon are recognized by a group which is not distinctly medical in its membership.—*New England Journal of Medicine,* October 30, 1930.